NON-PATENT LITERATURE

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File 155:MEDLINE(R) 1950-2007/Mar 15
File  73:EMBASE 1974-2007/Mar 20
File   5:Biosis Previews(R) 1926-2007/Mar W2
File  35:Dissertation Abs Online 1861-2007/Feb
File 156:ToxFile 1965-2007/Mar W2
File  45:EMCare 2007/Mar W2
File 144:Pascal 1973-2007/Mar W2
File  94:JICST-EPlus 1985-2007/Mar W4
Set     Items   Description
S1        340   ((CONTRAST OR RADIOCONSTRAST)()NEPHROPATHY)(S)(TREAT? OR P-
            REVENT?)
S2     848086   (BLOOD OR VEIN OR VENAL)(2N)PRESSURE
S3         20   S1 AND S2
S4          7   RD  (unique items)
S5          7   Sort S4/ALL/PY,A [not relevant]

[File 155] MEDLINE(R) 1950-2007/Mar 15
[File 5] Biosis Previews(R) 1926-2007/Mar W2
[File 73] EMBASE 1974-2007/Mar 20
[File 94] JICST-EPlus 1985-2007/Mar W4
[File 144] Pascal 1973-2007/Mar W2
[File 35] Dissertation Abs Online 1861-2007/Feb
[File 65] Inside Conferences 1993-2007/Mar 20
Set     Items   Description
S1      22978   S VEIN? ?(2N)(KIDNEY? ? OR RENAL)
S2    2813670   S OCCLUD? OR OCCLUSI??? OR BALLOON? ? OR BLOCK??? OR OBSTRUCT?
S3     748680   S BLOOD()PRESSURE
S4   10242608   S INCREAS??? OR ELEVAT??? OR RAIS? OR RISE? ?
S5   11362407   S LOWER??? OR DECREAS??? OR REDUC???? OR DECLIN? OR DROP
S6    1877186   S CONTRAST OR RADIOCONTRAST OR RADIOPAQUE
S7        674   S S2(2W)S1
S8      80518   S S3(2N)S4
S9      97690   S S3(2N)S5
S10         1   S S7 AND S8 AND S9 [not relevant]
S11       168   S S1(S)S2(S)3(S)S4(S)S5
S12       168   S S11 NOT S10
S13        22   S S6 AND S12
S14         9   RD  (unique items)
S15         9   SORT S14/ALL/PY,A
S16    510197   S NEPHROPATHY OR (KIDNEY? ? OR RENAL)(S)(DYSFUNCTION OR FUNCTION??? OR
INSUFFICIENCY OR INSULT)
S17       146   S S12 NOT S13
S18        57   S S17 AND S16
S19        26   RD  (unique items)
S20         0   S S19/2004:2005
S21         2   S S19/2006:2007
S22        24   S S19 NOT S21
S23        24   SORT S22/ALL/PY,A
S24        21   S S3(3N)S4:S5 AND S11
S25        18   S S24 NOT (S10 OR S13 OR S18)
S26        10   RD  (unique items)
S27        10   SORT S26/ALL/PY,A
S28       357   S PRESSURE(3N)S1
S29        47   S S1(10N)S2 AND S28
S30        42   S S29 NOT (S24 OR S10 OR S13 OR S18)
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S31 34 RD (unique items)
S32 0 S S31/2004:2005
S33 1 S S31/2006:2007
S34 33 S S31 NOT S33
S35 33 SORT S34/ALL/PY,A
S36 8 S S1(S)S2(S)S28 AND S16/TI,DE
S37 0 S S36 NOT (S29 OR S24 OR S10 OR S13 OR S18)

15/7/3 (Item 3 from file: 155)
Fulltext available through: ScienceDirect (Elsevier) USPTO Full Text Retrieval Options
MEDLINE(R)

05580682 **PMID:** 7276433
Renal chemoreceptors.
Recordati G; Moss N G; Genovesi S; Rogenes P
Journal of the autonomic nervous system (NETHERLANDS) Apr 1981 , 3 (2-4) p237-51 ,
ISSN: 0165-1838--Print **Journal Code:** 8003419
Contract/Grant No.: HL 02334; HL; NHLBI; NS 11132; NS; NINDS; T32 AM07047; AM; NIADDK
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
A study of the **renal** receptors and types of stimuli which give origin to supraspinal and spinal-mediated autonomic reflexes is presented. Multiunit and single unit recordings from the afferent **renal** nerves of male Sprague-Dawley rats have revealed two groups of **renal** chemosensitive receptors (chemoreceptors). These we have called **renal** R1 and R2 "chemoceptive" receptors. R1 receptors do not have a resting discharge but are activated after 38.7 +/- **3.3** (S.E) sec (n = 40) of complete **renal** ischemia (**occlusion** of the **renal** artery). Other activating stimuli are associated with a marked impairment in **renal** blood flow (prolonged **occlusion** of the **renal vein** and the hypotension of systemic asphyxia or hemorrhage). Their discharge is characterized by trains of impulses which cease abruptly upon re-entry of blood into the **kidney**. They are not responsive to **increases** or **decreases** in **renal** perfusion **pressure** or to **increases** in **renal** venous or ureteral **pressure**. In **contrast**, R2 receptors have a resting discharge and respond vigorously to backflow of normal urine (nondiuretic) into the **renal** pelvis. The results of the backflow into the pelvis of different test solutions (diuretic and nondiuretic urine, 1 M urea, 1 M mannitol and solutions of NaCl and KCl) indicate that this response is dependent upon the composition of the fluid bathing the **renal** pelvis rather than the **increase** in pelvic **pressure** or pelvic distension. The resting discharge rate is highest in nondiuretic conditions and **declines** substantially after diuresis is induced by extracellular volume expansion. R2 receptors are also activated by **renal** ischemia produced by clamping the **renal** artery. It is concluded that these two groups of afferent sensory units are **renal** chemosensitive receptors, (chemoreceptors) which respond to the chemical environment of **renal** interstitium.
Record Date Created: 19811118
Record Date Completed: 19811118

15/7/4 (Item 4 from file: 35)
Dissertation Abs Online

01383607 ORDER NO: AAD94-26809
CHARACTERIZATION OF THE VASOACTIVITY OF TACHYKININS IN ISOLATED RAT KIDNEY: FUNCTIONAL STUDIES AND IN VITRO RECEPTOR AUTORADIOGRAPHY
Author: CHEN, YUEJIN

Degree: PH.D.
Year: 1994
Corporate Source/Institution: EAST TENNESSEE STATE UNIVERSITY (0069)
Chairman: DONALD HOOVER
Source: Volume 5507B of Dissertations Abstracts International.
PAGE 2658 . 109 PAGES

Although tachykinins have potent vascular actions, their effect on **renal** resistance blood vessels is currently unknown.

The vasoactive properties of tachykinins and related analogs were assessed in isolated perfused rat **kidney**. At a basal perfusion **pressure** (PP) of 75 $\pm$ 6 mm Hg (n = 5), bolus injections of substance P (SP) had no significant vasoactive effect. Following a sustained **increase** in baseline PP (134 $\pm$ 10 mm Hg) produced by phenylephrine (1 μM), SP evoked a dose-dependent **increase** in PP. The largest dose of SP **increased** PP by 60 $\pm$ 5 mm Hg. The vasoconstrictor response to SP was not **blocked** by phentolamine when angiotensin II was used to **increase** basal tone. Thus, the response to SP is not mediated by norepinephrine. Pressor responses to SP were not potentiated by peptidase inhibitors, captopril and thiorphan. SP(1-7) had no effect on PP, suggesting that the pressor response to SP is C-terminal dependent and tachykinin receptor mediated. The selective NK-1 receptor agonist, (Sar$\sp9$,Met(O$\sb2)\sp{11}\rbrack$SP, had no effect on PP. In **contrast**, both the selective NK-2 and NK-**3** receptor agonists, GR-64349 and (MePhe$\sp7$) NKB, produced dose-dependent pressor responses (116 $\pm$ 8 and 134 $\pm$ 15 mm Hg **increases** in PP at 33 nmol, respectively) and were more potent than SP. Infusion of capsaicin (500 nM) produced an initial **increase** in PP following by a more prolonged **decrease** in PP. Clamping the **renal vein** produced a marked **increase** in PP.

The localization of NK-3 receptors in rat **kidney** evaluated by film autoradiography using $\sp{125}$I- (MePhe$\sp7\rbrack$NKB revealed a high density of specific binding sites on the proximal ureter and **renal** pelvis, moderate density in the **renal vein** and its large branches, and a low density in the inner strip of outer medulla, but no specific binding on the **renal** artery system and cortex. High resolution autoradiograms demonstrated $\sp{125}$I- (MePhe$\sp7\rbrack$NKB binding sites on the tunica media of the **renal vein** and tunica muscular**ise**s of **renal** pelvis and ureter. Specific binding of $\sp{125}$I-BHSP was found in association with the **renal** artery and **renal** pelvis. No specific SP binding sites were associated with **renal vein**.

These data indicate that the pressor effect of tachykinins in the isolated rat **kidney** can be mediated by NK-2 and/or NK-3 receptors. The latter may be on the vascular smooth muscle of the **renal vein**.

23/7/1 (Item 1 from file: 5)
Biosis Previews(R)

0000796908 **Biosis No.:** 19542800020663
Electrolyte and water excretions and renal hemodynamics during induced congestion of the superior and inferior vena cava of man
Author: FARBER SAUL J; BECKER WILLIAM H; EICHNA LUDWIGW
Author Address: New York U. Med., N. Y. C.
Journal: JOUR CLIN INVEST 32 ((11)): p 1145-1162 1953 1953
Document Type: Article
Record Type: Abstract
Language: Unspecified
Abstract: **Elevat**ed vena caval **pressure**s, ranging from 100 to 250 mm. saline soln., were produced for periods of 30 min. by the inflation of a **balloon** in the inferior vena cava above the **renal veins** in 16 subjects, in the inferior vena cava below the **renal veins** in 13, and in the superior vena cava in 12. During the venous congestion of each of the **3** areas essentially similar changes occurred in water and electrolyte excretions, **renal** hemodynamics, and arterial **blood pressure**s. The urinary excretions of Na and chloride, and less consistently of K and water, **decreased**. Urinary electrolyte concns. tended to

remain uncnanged, and the **reduced** electrolyte excretions were usually due to the **reduced** water excretion. Occasionally water excretions **decreased** little or not at all, yet electrolyte concn. fell decidedly. **Renal** plasma flow and glomerular filtration rate usually **decreased** by 15-25% at the onset of the venous congestion and concomitantly with the **reduction** in water and electrolyte excretions. As venous congestion was maintained, both **renal** hemodynamic **functions** improved and were returning toward control values while water and electrolyte excretions remained at their low levels or **decreased** further. Filtration fraction did not change. Systemic arterial **pressure** changed little; systolic **pressure** and pulse **pressure** fell slightly (5-10 mm. Hg), while diastolic **pressure** and mean **pressure** remained essentially unchanged. Heart rate and the ecg. remained unaltered. Arterial hematocrit and plasma protein concn. did not change. Following release of inferior vena caval congestion, both including and excluding the **kidneys**, water, and electrolyte excretions, **renal** plasma flow and glomerular filtration rate returned to, or well toward, control levels, the **renal** hemodynamic **functions** promptly, the urinary excretions within 30 min. Following release of superior vena caval congestion, water and electrolyte excretions returned toward control levels slowly, or not at all, and were still **reduced** 30 min. after release of the congestion. **Renal** plasma flow and glomerular filtration rate, on the other hand, returned promptly to control values. These observations indicate that acute congestion of a sizable segment of the venous system induces a **decreased** urinary excretion of water and electrolytes. The mechanisms responsible for this effect remain to be detd. ABSTRACT AUTHORS: Authors

23/7/3 (Item 3 from file: 5)
Fulltext available through: USPTO Full Text Retrieval Options
Biosis Previews(R)

05928132 **Biosis No.**: 198069042119
RENAL FUNCTION CORTICAL BLOOD FLOW AND MORPHOMETRY IN ISCHEMIC ACUTE RENAL FAILURE IN THE RAT
Author: AXELSEN R A (Reprint); CARTWRIGHT V E
Author Address: DEP PATHOL, MED SCH, UNIV QUEENSL, HERSTON RD, HERSTON, QUEENSL 4006, AUST **AUSTRALIA
Journal: Pathology 11 (4): p 629-640 1979
ISSN: 0031-3025
Document Type: Article
Record Type: Abstract
Language: ENGLISH
Abstract: Unilateral post-ischemic acute **renal** failure (ARF) was produced in rats by **occluding** the left **renal** artery and **vein** for 1 h. Left **renal function** was assessed 1 or 2 h after the end of the period of ischemia and the **kidneys** fixed by arterial perfusion. ARF was characterized by **increased** urine flow (8.1 .+-. 1.2 SEM .mu.l/min per 100 g body wt, n = 14; controls 1.0 .+-. 0.1, n = 11), **decreased** urinary osmolality (335 .+-. 12 m osm/kg, n = 14; controls 1885 .+-. 97, n = 11), and markedly **reduced** 3H inulin urine/plasma ratio (**3**.98 .+-. 0.56, n = 14; controls 499 .+-. 60, n = 9) and 3H inulin clearance (32.9 .+-. 7.0 .mu.l/min per 100 g body wt, n = 14; controls 505 .+-. 45, n = 9). **Renal** cortical blood flow, determined by the hydrogen desaturation technique, was less in animals with ARF (4.2 .+-. 0.4 ml/min per ml of cortex, n = 8) than in controls (5.4 .+-. 0.6, n = 5), but not significantly so. In vivo stereomicroscopic examination of the left **renal** surface in ARF revealed dilated proximal convoluted tubules and delayed passage of i.v. injected dye (lissamine green) through these tubules. Histological examination also showed dilated proximal convoluted tubules and cellular debris impacted in the terminal straight portions of proximal tubules and thin limbs of the loops of Henle. Light microscopic morphometric studies demonstrated significant dilatation of proximal convoluted tubules and Bowman's spaces, and significant narrowing of the lumina of distal convoluted tubules and cortical collecting ducts. Tubular **obstruction** in a polyuric model of ischemic ARF is significant in the absence of a marked **decrease** in

renal cortical blood flow. Morphometric studies are of value in experimental ARF. The usefulness of the hydrogen desaturation technique for the determination of **renal** cortical blood flow, reported herein for the 1st time in ischemic ARF, is emphasized.

23/7/10 (Item 10 from file: 155)
Fulltext available through: USPTO Full Text Retrieval Options
MEDLINE(R)

08494511 **PMID**: 2194031
Effects of nephrectomy on hypertension, renin activity and total renal function in patients with chronic renal artery occlusion.
Sonkodi S; Abraham G; Mohacsi G
First Department of Internal Medicine, Albert Szent-Gyorgyi Medical University, Szeged, Hungary.
Journal of human hypertension (ENGLAND) Jun 1990 , 4 (3) p277-9 , **ISSN**: 0950-9240--Print **Journal Code**: 8811625
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Eight hypertensive patients (5 men and 3 women, aged between 31 and 64 years) with chronic total **occlusion** of one **renal** artery were observed for the past ten years. The peripheral plasma renin activity (PRA) and the **renal vein** PRA ratio (mean 3.18) were abnormally high in all cases. Because of the small size of the affected **kidney** (less than 9 cm in length), nephrectomy was the chosen treatment. Postoperative investigations revealed **decreases** in **blood pressure** (from 202/118 to 147/93 mmHg), peripheral PRA (from 6.05 to 1.05 ng/ml/h; P less than 0.001), serum creatinine (from 188.8 to 145.1 mumol/100 ml) urine volume (from 1937.5 to 1214.**3** ml) and **increases** in endogenous creatinine clearance (from 36.57 to 53.0 ml/min). The results suggest that, apart from the **decrease** in **blood pressure**, the nephrectomy led to the disappearance of a factor which depresses the **renal function** in cases of chronic **renal** artery **occlusion** and which may be related to the renin-angiotensin system.
Record Date Created: 19900808
Record Date Completed: 19900808

23/7/16 (Item 16 from file: 5)
Fulltext available through: USPTO Full Text Retrieval Options
Biosis Previews(R)

12918087 **Biosis No.**: 199598385920
Renovascular hypertension new diagnostic and therapeutic procedures
Author: Jensen Gert
Author Address: Dep. Nephrol., Sahlgrenska Univ. Hosp., Univ. Goteborg, S-413 45 Goteborg, Sweden**Sweden
Journal: Scandinavian Journal of Urology and Nephrology Supplementum 0 (170): p 2-78 1995 1995
ISSN: 0300-8886
Document Type: Article
Record Type: Abstract
Language: English
Abstract: Renovascular hypertension (RVH) remains a leading cause of potentially curable hypertension, Although RVH affects less than 1% of the unselected hypertensive population, between 10% and 35% of appropriately screened patients referred to specialised centres for problematic hypertension may prove to have renovascular disease.

Advances in percutaneous transluminal **renal** angioplasty (PTRA) have renewed interest in developing better noninvasive screening tests for identifying patients with potentially correctable hypertension or **renal** impairment due to renovascular disease caused by either fibromuscular dysplasia (FMD) or arteriosclerosis. Duplex ultrasound with the two-dimensional Echo-Colour-Doppler technique for measurements of blood flow velocities in the **renal** interlobar arteries as expressed in the Pulsatility Index (PI) has been evaluated. Experimentally induced changes in renovascular resistance (RVR) in normotensives and in primary hypertensives were registered noninvasively by means of PI-measurements. A significant correlation between the absolute values of PI and RVR was found in hypertensives (r = 0.50, p lt 0.002), but not in normotensives. In both groups, the changes of RVR due to angiotensin II infusion and ACE-inhibition were significantly correlated to the changes in PI (normnotensives: r = 0.69, p lt 0.001, primary hypertensives : r = 0.64, p lt 0.001). Normally, the blood flow velocities as expressed by the PI in the **renal** vasculature of the two **kidneys** are equal. In hypertensive patients, PI was **lower** in **kidneys** with significant **renal** artery stenosis (RAS) than in **kidneys** without RAS (p lt 0.001). Doppler signals were absent in all **kidneys** with **renal** artery **occlusion**. A bilateral low PI combined with normal side difference in PI may in hypertensive patients indicate bilateral RAS. RVH was correctly diagnosed in 84% of the patients and the presence of RAS in 94%. Provocative testing of an activated renin-angiotensin system by means of an angiotensin converting enzyme inhibitor (ACEI) constitutes the foundation for screening for RVH using gamma camera renography with 99mTc-DTPA as a glomerular filtration marker. In 20 consecutive patients with successfully treated RVH, one-third of the patients were not correctly diagnosed using ACEI-enhanced 99mTc-DTPA gamma camera renography, which indicates that some patients with RVH have compensatory mechanisms to maintain GFR after ACE inhibition. The relationship between the renin-angiotensin system and erythropoietin (EPO) production was studied in 20 patients with RAS and hypertension. Higher EPO levels were found in patients with a unilaterally activated renin-angiotensin system compared with patients without (27.3+-16.8 mU/ml vs. 14.1+-11.3 mU/ml, p lt 0.05), but no **increase** in **renal vein** EPO concentration from the **functional** stenotic **kidney** was found. EPO measurements in **renal vein** or peripheral blood cannot be used in the diagnostic work-up in patients with suspected **renal** artery stenosis. Percutaneous transluminal **renal** angioplasty (PTRA) was performed in 180 **renal** arteries in 137 patients, 30 of which had FMD and 107 arteriosclerotic vascular disease (AVD). The cure and improvement rate for hypertension at one year's follow-up was 86% in FMD and 64% in AVD patients. Comparing dynamic **renal vein** renin release studies with clinical outcome, a sensitivity of 95% and a specificity of 75% was achieved. Total **renal function** was improved in both groups (p lt 0.001), the improvement being made by the revascularised **kidney**. The incidence of' complications was 5%. The angiographic one-year followup revealed a recurrence rate of 6.7% for FMD and 15.1% for AVD patients. PTRA may be considered firmly established as the method of first choice for treatment of RVH.

27/7/5 (Item 5 from file: 155)
Fulltext available through: custom link USPTO Full Text Retrieval Options
MEDLINE(R)

08758100 **PMID**: 1992825
Responses of spinoreticular cells to graded increases in renal venous pressure.
Ammons W S
Department of Physiology, Thomas Jefferson University, Philadelphia, Pennsylvania 19107.
American journal of physiology (UNITED STATES) Jan 1991 , 260 (1 Pt 2) pR27-31 ,
ISSN: 0002-9513--Print **Journal Code**: 0370511
Contract/Grant No.: HL-36378; HL; NHLBI
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: MEDLINE; Completed
Previous work established that **occlusion** of the **renal vein** excites spinoreticular tract (SRT) neurons of the cat. The present study was designed to determine the relationship between **renal vein pressure** level and SRT cell activity. Experiments were performed on 40 cats that were anesthetized with alpha-chloralose. Sixty SRT neurons in the T12-L2 segments were tested for responses to a **renal vein pressure** (RVP) of 60 mmHg. Twenty-three cells responded with an **increase** in activity. Stimulus response relationship for these cells were determined over the RVP range of 10-80 mmHg. RVP thresholds averaged 25 +/- 3 mmHg. Above this level greater **increases** in RVP were associated with greater **increases** in neuronal activity. At RVP of 80 mmHg cell activity **increased** from 4 +/- 2 to 21 +/- 5 spikes/s. Cells with both A delta- and C-fiber **renal** afferent inputs had significantly greater responses and **lower** thresholds than cells with only A delta input. **Renal vein occlusion evoked increases in blood pressure.** At a **renal vein pressure** of 80 mmHg, pressor responses averaged 16 +/- 5 mmHg. No significant changes in heart rate were observed. The results demonstrate that SRT cells are capable of encoding the level of RVP. Such responses may be important for grading of reflexes of **renal** origin that require supraspinal circuitry.
Record Date Created: 19910312
Record Date Completed: 19910312

35/7/1 (Item 1 from file: 5)
Biosis Previews(R)

0000700515 **Biosis No.:** 19512500033487
Effect of renal venous occlusion on intrarenal pressure
Author: SWANN H G; MONTGOMERY A V; LOWRY J S
Author Address: U. Texas Med. Br., Galveston
Journal: PROC SOC EXPTL BIOL AND MED 76 ((4)): p 773-777 1951 1951
Document Type: Article
Record Type: Abstract
Language: Unspecified
Abstract: When the **renal vein** is completely **occluded**, the intra**renal pressure increases** about 3-fold, being **elevated** to the simultaneous **pressure** in the **vein** on the **renal** side of the **occlusion**. Usually this **pressure** is somewhat less than the simultaneous diastolic **blood pressure**. When the **vein** is partially **occluded**, intra**renal pressure** does not change until the simultaneous **pressure** in the **vein** on the **renal** side of the **occlusion rises** to the original intra**renal pressure**. At this point, as the **occlusion** is **increased**, the 2 **pressures rise** together and always have the same value. The hypothesis is proposed that the **pressure** inside the **renal** peritubular capillaries, venules, uriniferous tubules and lymphatics is normally just above intra**renal pressure**, i.e., at 25 mm. Hg. The authors suppose that just as these vessels leave the **renal** parenchyma the **pressure** in them abruptly **declines** toward zero, and this situation permits reabsorption to take place, freed from the influence of random or pathological fluctuations in vena cava **pressure**. Oncotic **pressures** and cellular metabolic processes, therefore, are presumed to be the primary determinants of differential reabsorption. ABSTRACT AUTHORS: H. G. Swann

35/7/2 (Item 2 from file: 155)
Fulltext available through: USPTO Full Text Retrieval Options
MEDLINE(R)

00933679 **PMID:** 13529697 **Record Identifier:** 5834-22169-323-326-491
The effect of increasing pressure in the renal veins and of obstruction to renal lymphatic outflow upon urinary protein concentration.
KLAUS R; SHALLOW J; MURPHY J J

Surgical forum (Not Available) 1957 , 8 p613-6 , **ISSN:** 0071-8041--Print **Journal Code:** 0337723
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Other Citation Owner: CLML
Record type: OLDMEDLINE; Completed
Record Date Created: 19581201
Record Date Completed: 20000701

35/7/4 (Item 4 from file: 5)
Biosis Previews(R)

0000976315 **Biosis No.:** 19593300021318
Elevation of inferior vena cava pressure and thoracic lymph and urine flow
Author: KATZ YALE J; COCKETT A T K
Author Address: U. Southern California Sch. Med., Los Angeles
Journal: CIRCULATION RES 7 ((1)): p 118-122 1959 1959
Document Type: Article
Record Type: Abstract
Language: Unspecified
Abstract: With an **increase** in **renal vein pressure** by partial **obstruction** of the inferior vena cava in dogs, there follows an **increase** in thoracic lymph flow and a simultaneous **increase** in urine flow and urine sodium excretion per minute. The **increase** in lymph flow appears to be caused by an **increased** production of **renal** lymph, since control animals with absent or non**functioning** **kidneys** fail to show this lymph flow **increase**. **Renal** deviation of fluid and sodium to the lymphatics may similarly occur in heart failure and account for the retention of sodium and water. ABSTRACT AUTHORS: Authors

35/7/8 (Item 8 from file: 5)
Biosis Previews(R)

0001642620 **Biosis No.:** 19664700046721
Histologic and functional changes following partial occlusion of the renal vein in the dog: A preliminary study
Author: SANDROLINI JAMES A; TORRES CESAR; POLLAK VICTOR E
Author Address: Presbyterian-Saint Luke's Hosp., Chicago, Ill., USA
Journal: INVEST UROL 3 ((1)): p 83-91 1965 1965
Document Type: Article
Record Type: Abstract
Language: Unspecified
Abstract: An experimental model was utilized to study **renal** physiologic and morphologic changes in the dog following unilateral partial **renal vein** ligation. An **increased** **renal** venous **pressure**, to between 100 and 300 mm of saline, was not associated with significant proteinuria from either the affected or the control **kidney**. Histologic changes occurred following **renal vein** ligation. The most striking of these were **increased** protein **droplets** in the tubular cells, leukocyte margination in the glomerular capillaries, and some thickening of the glomerular basement membrane. The significance of the **functional** and histologic findings is discussed. Further studies are needed before conclusions are drawn concerning the relationship of **renal vein pressure** to proteinuria and morphologic changes. ABSTRACT AUTHORS: Authors

35/7/9 (Item 9 from file: 5)

Biosis Previews(R)

0001945871 **Biosis No.**: 19684900104512
Mechanism of the reflex depressor effect by kidney in dog
Author: UEDA HIDEO; UCHIDA YASUMI; KAMISAKA KAZUAKI
Author Address: Fac. Med.. Univ., Tokyo, Jap.
Journal: JAP HEART J 8 ((6)): p 597-606 1967 1967
Conference/Meeting: XXXIII International Congress of Physiol-logical Sciences: First International Symposium on Pulmonary Edema, Tokyo, Jap., September 2, 1965
Document Type: Meeting
Record Type: Abstract
Language: Unspecified
Abstract: The mechanism ot the reflex depressor **function** from the **kidney** was studied in anesthetized dogs with pentobarbitone sodium under artificial breathing. The action potentials of the efferent **renal** nerve (sympathetic) were **decreased** by compression of the **kidney** and **renal vein occlusion**, and were followed by a fall in systemic arterial **pressure**. The efferent action potentials were slightly **increased** or unchanged by **renal** artery **occlusion**, but were followed by a **rise** in the systemic arterial **pressure**. The action potentials of the afferent **renal** nerve **increased** with the **rise** in the intra**renal pressure** produced by compression of the **kidney**, **renal vein occlusion** , and **elevation** of the perfusion **pressure** of the **kidney** in cross-perfusion experiments. The afferent discharge **decreased** with the fall in the intra**renal pressure** produced by **renal** artery **occlusion**. The action potentials of the efferent nerve **decreased** by electric stimulation of the afferent nerve, and were followed by a fall in the systemic arterial **pressure**. The action potentials of the afferent nerve evoked by compression of the **kidney**, **elevation** of the intrapelvic **pressure**, and **renal vein occlusion**, did not vanish after subcapsular and intrapelvic injection of 2% procaine or total decapsulation. The evoked action potentials disappeared after injection of procaine into the **renal** artery. The receptor, which is sensitive to changes of intra**renal pressure**, is activatec by **elevation** of the intra**renal pressure** and causes reflex hypotension by means of the inhibition of the sympathetic discharge. ABSTRACT AUTHORS: Authors

35/7/10 (Item 10 from file: 155)
Fulltext available through: USPTO Full Text Retrieval Options
MEDLINE(R)

02702466 **PMID**: 5359478
Experimental obstructive nephropathy in the pig. II. Pathology.
Matz L R; Craven J D; Hodson C J
British journal of urology (ENGLAND) Dec 1969 , 41 pSuppl:21-35 , **ISSN**: 0007-1331--Print **Journal Code**: 15740090R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Record Date Created: 19700202
Record Date Completed: 19700202

35/7/11 (Item 11 from file: 155)
Fulltext available through: USPTO Full Text Retrieval Options ProQuest
MEDLINE(R)

02599954 **PMID**: 5770214
Left renal vein pressure following acute occlusion in man.

Killen D A; Zukoski C F; Edwards R H
American surgeon (UNITED STATES) Jun 1969 , 35 (6) p439-43 , **ISSN:** 0003-1348--Print **Journal Code:** 0370522
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Record Date Created: 19690703
Record Date Completed: 19690703

35/7/29 (Item 29 from file: 94)
Fulltext available through: USPTO Full Text Retrieval Options
JICST-EPlus

01877568 **JICST Accession Number:** 93A0721266 **File Segment:** JICST-E
The influence of increased venous pressure on renal circulation and function. The role of high right atrial pressure on renal insufficiency during left univentricular support.
AKAMATSU HIDEKI (1); ARAI HIROKUNI (1); SAKAMOTO TOORU (1); SUZUKI AKIO (1)
(1) Tokyo Medical and Dental Univ.
Jinko Zoki, Nippon Jinko Zoki Gakkai (Japanese Journal of Artificial Organs) , 1993 , VOL.22,NO.4 , PAGE.1193-1198 , FIG.9, REF.10
Journal Number: Z0557BAD **ISSN:** 0300-0818
Universal Decimal Classification: 616/618-76/78
Language: Japanese **Country of Publication:** Japan
Document Type: Journal
Article Type: Original paper
Media Type: Printed Publication
Abstract: Although clinical experience showed the effectiveness of LVAD for maintaining systemic flow and **pressure** on postcardiotomy cardiogenic shock patients, **renal insufficiency** might likely progress when the right atrial **pressure**(RAP) was high. The influence of **increase**d venous **pressure** on **renal** circulation and **function** was studied using eight adult mongrel dogs. An **occlusion balloon** catheter was applied selectively in left **renal vein** to obtain high **renal vein pressure**(RVP), simulating clinical settings of high RAP with normal **blood pressure**(BP) and cardiac output(CO). **Renal** blood flow, urine output and free water clearance of left **kidney**(RVP=25.1.+-.1.6mmHg) were significantly **lower** than those of right **kidney**(RVP=2.6.+-.0.7mmHg), despite of normal BP and CO. To avoid the progression of **renal insufficiency**, early application of RVAD would be useful for the patient who in supported by LVAD and has high RAP. (author abst.)

[File 9] **Business & Industry(R)** Jul/1994-2007/Mar 19
[File 16] **Gale Group PROMT(R)** 1990-2007/Mar 19
[File 160] **Gale Group PROMT(R)** 1972-1989
[File 149] **TGG Health&Wellness DB(SM)** 1976-2007/Mar W1
[File 148] **Gale Group Trade & Industry DB** 1976-2007/Mar 09
[File 135] **NewsRx Weekly Reports** 1995-2007/Mar W2
[File 441] **ESPICOM Pharm&Med DEVICE NEWS** 2007/Sep W2
[File 636] **Gale Group Newsletter DB(TM)** 1987-2007/Mar 19

Set	Items	Description
S1	426	S VEIN? ?(2N)(KIDNEY? ? OR RENAL)
S2	1251033	S OCCLUD? OR OCCLUSI??? OR BALLOON? ? OR BLOCK??? OR OBSTRUCT?
S3	101581	S BLOOD()PRESSURE
S4	10253033	S INCREAS??? OR ELEVAT??? OR RAIS? OR RISE? ?
S5	8183481	S LOWER??? OR DECREAS??? OR REDUC???? OR DECLIN? OR DROP
S6	502751	S CONTRAST OR RADIOCONTRAST OR RADIOPAQUE
S7	1147952	S PRESSURE
S8	33956	S NEPHROPATHY OR (KIDNEY? ? OR RENAL)(S)(DYSFUNCTION OR FUNCTION??? OR INSUFFICIENCY OR INSULT)
S9	19	S S1(3N)S2
S10	12667	S S3(2N)S4
S11	18193	S S3(2N)S5
S12	0	S S9(S)S10(S)S11
S13	6	S S1(S)S2(S)S3
S14	**4**	**RD (unique items)**
S15	78	S S8(S)S1
S16	4	S S2(S)S7(S)S15
S17	**2**	**S S16 NOT** S13 [not relevant]

14/3,K/2 (Item 2 from file: 149)
TGG Health&Wellness DB(SM)

01473323 **Supplier Number**: 14825526 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Molecular biology of the renin cascade in hypertension.
Weston, Philip; Swales, John D.; Thurston, Herbert; Lodwick, David; Samani, Nilesh
The Lancet , v343 , n8890 , p151(4)
Jan 15 , 1994
Publication Format: Magazine/Journal
ISSN: 0099-5355
Language: English
Record Type: Fulltext; Abstract **Target Audience**: Professional
Word Count: 4002 **Line Count**: 00331
...the rat, **renal** artery constriction plus contralateral nephrectomy produced a rapid and severe **rise** in **blood pressure** with evidence of volume overload.[4] The renin content of the clipped **kidney dropped** when...**renal** venous renin rose only when the opposite **kidney** was left in situ.[6] Moreover, **blockade** of the renin-angiotensin system by the angiotensin II antagonist saralasin **lowered** the **blood pressure** only of 2K1C hypertensive rats.[7] Total exchangeable sodium levels rose in rats with contralateral...
...hypertension in the 1K1C model since dietary salt restriction does not prevent the **rise** in **blood pressure** after clipping.[9] In the 2K1C model there is a direct relation between plasma renin concentration and **blood pressure** in the early phase of hypertension (less than 6 weeks after **renal** artery constriction) but...
...chronic phase (more than 4 months after clipping). Moreover, in the chronic phase renin-angiotensin **blockade** by lengthy infusion of saralasin or angiotensin-coverting-enzyme (ACE) inhibitor does not **lower**

the **blood pressure**.[10] Thus, the renin-angiotensin system seems to be far less important for the maintenance of **blood pressure** in chronic 2K1C hypertension. However, removal of the **renal** artery clip restores **blood pressure** rapidly to normal in the 2K1C and 1K1C models with long-standing hypertension.[10,11] In the 1K1C model, removal of the clip led to natriuresis and a fall in **blood pressure**, whereas in the 2K1C model a modest degree of sodium retention occurred.[11] Thus in...
...response to unclipping. In the 1K1C model plasma renin is low normal, yet unclipping restores **blood pressure** to normal. In translating these results to human renovascular hypertension, several parallels can be drawn. In the 2K1C situation neither plasma renin nor plasma **renal vein** renin concentrations can be predictors of success for reconstructive surgery or angioplasty. The patient described...

14/3,K/3 (Item 3 from file: 149)
TGG Health&Wellness DB(SM)

01281788 **Supplier Number**: 10842121
Chronic pyelonephritis: the significance of renal renin and the vascular changes in the human kidney.
Peh, Suat Cheng; Lindop, George B.M.
Journal of Pathology , v163 , n4 , p343(7)
April , 1991
Publication Format: Magazine/Journal
ISSN: 0022-3417
Language: English
Record Type: Abstract **Target Audience**: Professional
Abstract: ...bacterial infection which, in both its acute and chronic forms, may be associated with high **blood pressure** (hypertension). The walls of the **renal** artery (which delivers blood to the **kidney**) in pyelonephritis ... ...juxtaglomerular apparatus (JGA), a group of cells in the **kidney** critical for the regulation of **blood pressure**. The JGA produces renin, which causes vasoconstriction (constriction of blood vessels, **raising** **blood pressure**). Renin-containing cells (RCCs) are known to be abnormally distributed in some **kidney** diseases. To... ...surgically removed from patients with that disease were examined. Six of the patients had high **blood pressure** and 12 had normal **blood pressure**. Five of the six **kidneys** from patients with high **blood pressure** and seven of the 12 **kidneys** from patients with normal **blood pressure** had more than the normal number of RCCs, with no differences noted with respect to **blood pressure** in number or distribution of these cells. No relationships were found between the degree of narrowing and **blood pressure** or the degree of narrowing and the number of RCCs. **Kidney veins** were more severely damaged than **kidney** arteries, and many were **blocked** by clotted blood. These findings do not support an association between hypertension in pyelonephritis and narrowing of the arteries within the **kidney**. Extensive damage to **kidney veins** may impair the blood flow within the organ, leading to the tissue damage characteristic of...

File 149:TGG Health&Wellness DB(SM) 1976-2007/Mar W1
(c) 2007 The Gale Group

Set	Items	Description
S1	19	((CONTRAST OR RADIOCONSTRAST)()NEPHROPATHY)(S)(TREAT? OR P-REVENT?)
S2	7	S1/2004:2005
S3	1	S1/2006:2007
S4	11	S1 NOT S2:S3
S5	**11**	**Sort S4/ALL/PD,A**

5/3,K/2
DIALOG(R)File 149:TGG Health&Wellness DB(SM)
(c) 2007 The Gale Group. All rts. reserv.
01813480 SUPPLIER NUMBER: 53545095 (USE FORMAT 7 OR 9 FOR FULL TEXT)
PREPARED: PREParation for Angiography in REnal Dysfunction(*): A Randomized Trial of Inpatient vs Outpatient Hydration Protocols for Cardiac Catheterization in Mild-to-Moderate Renal Dysfunction.
Taylor, Allen J.; Hotchkiss, David; Morse, Robert W.; McCabe, John
Chest, 114, 6, 1570(1)
Dec, 1998
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-3692
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 3454 LINE COUNT: 00395
... to detect a difference between groups for a threshold change in creatinine considered to represent " **contrast nephropathy** ." However, the primary end point used in this study, the maximal change in creatinine 48 h postcatheterization, has been established as a relevant parameter for the comparison of protocols to **prevent** CRD.(1)
Clinical Implications
PREPARED suggests that an outpatient hydration protocol that consists of oral...

5/3,K/3
DIALOG(R)File 149:TGG Health&Wellness DB(SM)
(c) 2007 The Gale Group. All rts. reserv.
01838135 SUPPLIER NUMBER: 54825111 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Prophylaxis against contrast-induced nephropathy. (Commentary)
The Lancet, 353, 9165, 1638
May 15, 1999
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0099-5355
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 1200 LINE COUNT: 00105
... et al. A prospective randomized trial of prevention measures in patients at high risk for **contrast nephropathy** . J Am Coll Cardiol 1999; 33: 403-11.
7. Hans SS, Hans BA, Dhillon R...

FOREIGN AND INTERNATIONAL PATENTS

File 350:Derwent WPIX 1963-2006/UD=200719
File 347:JAPIO Dec 1976-2006/Nov(Updated 070228)

Set	Items	Description
S1	**7**	**((CONTRAST OR RADIOCONSTRAST)()NEPHROPATHY)(S)(TREAT? OR P-REVENT?) [not relevant]**

[File 350] **Derwent WPIX** 1963-2006/UD=200719
[File 347] **JAPIO** Dec 1976-2006/Nov(Updated 070228)

Set	Items	Description
S1	190	S VEIN? ?(2N)(KIDNEY? ? OR RENAL)
S2	1621031	S OCCLUD? OR OCCLUSI??? OR BALLOON? ? OR BLOCK??? OR OBSTRUCT?
S3	15453	S BLOOD()PRESSURE
S4	2914221	S INCREAS??? OR ELEVAT??? OR RAIS? OR RISE? ?
S5	6113776	S LOWER??? OR DECREAS??? OR REDUC???? OR DECLIN? OR DROP
S6	98562	S CONTRAST OR RADIOCONTRAST OR RADIOPAQUE
S7	1813190	S PRESSURE
S8	11982	S NEPHROPATHY OR (KIDNEY? ? OR RENAL)(S)(DYSFUNCTION OR FUNCTION??? OR INSUFFICIENCY OR INSULT)
S9	11	S S1(3N)S2
S10	1255	S S4(3N)S3
S11	3750	S S5(3N)S3
S12	**1**	**S S9 AND S10 AND S11 [a duplicate]**
S13	10	S S9 NOT S12
S14	2	S S3 AND S9
S15	**1**	**S S14 NOT S12 [not relevant]**
S16	9	S S13 NOT S14
S17	4	S S7 AND S9
S18	**2**	**S S17 NOT (S12 OR S14) [not relevant]**
S19	**7**	**S S9 NOT (S12 OR S14 OR S17)**
S20	26	S S1 AND S6
S21	4	S S3 AND S20
S22	**3**	**S S21 NOT (S9 OR S12 OR S14 OR S17) [1 duplicate; 2 not relevant]**
S23	276	S S6 AND S8
S24	12	S S1 AND S23
S25	**8**	**S S24 NOT (S21 OR S9 OR S12 OR S14 OR S17)**
S26	9	S S1(S)S2(S)S7
S27	**5**	**S S26 NOT (S24 OR S21 OR S9 OR S12 OR S14 OR S17) [not relevant]**

19/5/5 (Item 5 from file: 350)
Derwent WPIX

0010425149 *Drawing available*
WPI Acc no: 2001-023672/200103
Related WPI Acc No: 2002-061375
XRPX Acc No: N2001-018423
Pericardial procedure for inserting by=pass shunt into atrio-venus system during surgery, involves inflating balloon near renal veins to occlude flow of blood into retrohepatic vena cava
Patent Assignee: ANDERSON A A (ANDE-I); ANDERSON B B (ANDE-I)
Inventor: ANDERSON A A; ANDERSON B B
Patent Family (1 patents, 1 countries)

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 6148825	A	20001121	US 199823488	A	19980213	200103	B

Priority Applications (no., kind, date): US 199823488 A 19980213
Alerting Abstract US A
NOVELTY - A shunt is fixed within a venous system so that the distal portion of the shunt lies within an inferior vena cava (9). A **balloon** is inflated near **renal veins** (10) to **occlude** the flow of blood from **renal veins** and inferior vena cava into a retrohepatic vena cava so that the blood will be diverted into the vena caval opening of the shunt through the shunt and out of an atrial opening into a right atrium (8).
DESCRIPTION - An INDEPENDENT CLAIM is also included for an infra diaphragmatic procedure.
USE - For inserting by=pass shunt into atrio-venus system during surgery on retrohepatic vena cava.
ADVANTAGE - Eliminates need for customization of devices not originally intended for shunting the blood. Enables easy and accurate identification of the site within pericardium.
DESCRIPTION OF DRAWINGS - The figure shows the explanatory drawing of device **function**ally seated in venous system.
8 Right atrium
9 Inferior vena cava
10 **Renal veins**

19/5/7 (Item 7 from file: 350)
Derwent WPIX

0001124368
WPI Acc no: 1976-A8235X/197604
Vena cava inferior catheter - branch with inflatable cuff maintains shunt blood flow when occluding renal veins
Patent Assignee: MOSC MEDICINE INST (MOME-R)
Patent Family (1 patents, 1 countries)

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
SU 467743	A	19750514	SU 1895182	A	19730323	197604	B

Alerting Abstract SU A
The catheter features an elastic tuve (1) with apertures (2) on its side and inflatable **balloons** (3/4) separately connected to the tube for compression. The inflatable cuff (8) is blown through branch (7) for stopping the blood flow from the section in the vena cava while the blood flow from the **kidney** is carried out through apertures (2).

25/5/7 (Item 7 from file: 350)
Derwent WPIX

0010348690 *Drawing available*
WPI Acc no: 2000-664066/200064
XRAM Acc no: C2000-201093
XRPX Acc No: N2000-492074
Non-invasive measurement of renal hemodynamic functions using magnetic resonance imaging
Patent Assignee: UNIV CALIFORNIA (REGC)
Inventor: DUMOULIN C L; KATZBERG R W
Patent Family (1 patents, 1 countries)

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 6122540	A	20000919	US 1994257011	A	19940608	200064	B
			US 1995497024	A	19950630		

Priority Applications (no., kind, date): US 1994257011 A 19940608; US 1995497024 A 19950630
Alerting Abstract US A

NOVELTY - Measuring **renal function** in a living subject comprising obtaining a longitudinal spin relaxation time of the subject's blood, injecting a longitudinal spin relaxation time **contrast** agent, measuring a longitudinal spin relaxation time of pre- and postfiltered blood and calculating a filtration fraction, is new.

DESCRIPTION - A method of measuring **renal function** in a living subject comprises:

obtaining a longitudinal spin relaxation time of the subject's blood without any added **contrast** agents, T_1B;

injecting a longitudinal spin relaxation time **contrast** agent into the subject's blood shortening the longitudinal spin relaxation time of the blood;

measuring a longitudinal spin relaxation time of prefiltered blood, T_1A, prefiltered blood being blood on its way into the **kidney**, with magnetic resonance (MR) techniques, subsequent to injecting the **contrast** agent;

measuring a longitudinal spin relaxation time of postfiltered blood, T_1V, post-filtered blood being **blood** on its way out of the **kidney**, with magnetic resonance (MR) techniques, subsequent to injecting the **contrast** agent; and

calculating a filtration fraction, FF, from T_1A, T_1B and T_1V indicating the fraction of **contrast** agent filtered from the **subject**'s blood.

USE - The method is used to measure the glomerular filtration rate (GFR) from the filtration fraction (FF) and the **renal** plasma flow (RPF) with the formula GFR = FF x RPF.

INVENTOR

[File 350] **Derwent WPIX** 1963-2006/UD=200719
[File 347] **JAPIO** Dec 1976-2006/Nov(Updated 070228)

Set	Items	Description
S1	143	S AU=(GELFAND M? OR GELFAND, M?)
S2	124	S AU=(LEVIN H? OR LEVIN, H?)
S3	40	S S1 AND S2
S4	2	S RENAL()NEPHROPATHY
S5	0	S S1:S2 AND S4
S6	36972	S KIDNEY? ? OR RENAL
S7	24	S S3 AND S6
S8	53290	S OCCLUD? OR OCCLUS? OR BALLOON? ?
S9	20556	S VEIN? ? OR VENOUS
S10	**3**	**S S7 AND S8**
S11	4097	S INSULT OR NEPHROPATHY
S12	6	S S1:S2 AND S6 AND S11
S13	**4**	**S S12 NOT S10**
S14	15453	S BLOOD()PRESSURE
S15	2	S S1:S2 AND S6 AND S9 AND S14
S16	0	S S15 NOT (S10 OR S12)
S17	96732	S RADIOCONTRAST OR CONTRAST
S18	6	S S1:S2 AND S6 AND S17
S19	0	S S18 NOT (S10 OR S12)

10/26,TI/3 (Item 3 from file: 350)
Derwent WPIX

0010767341
WPI Acc no: 2001-381519/
Method of perfusion of kidney in patient having beating heart, using perfusion catheter assembly having introducer catheter and perfusion catheter tip

10/5/1 (Item 1 from file: 350)
Derwent WPIX

0014443757 *Drawing available*
WPI Acc no: 2004-634443/200461
Related WPI Acc No: 2004-614920
XRAM Acc no: C2004-227854
XRPX Acc No: N2004-501534
Protecting kidney in patient from insult involves reducing renal vein blood pressure from elevated blood pressure
Patent Assignee: PLC SYSTEMS INC (PLCS-N)
Inventor: **GELFAND M; LEVIN H; LEVIN H R**
Patent Family (3 patents, 107 countries)

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20040167415	A1	20040826	US 2003449174	P	20030224	200461	B
			US 2003449263	P	20030224		
			US 2004784231	A	20040224		
WO 2004075776	A2	20040910	WO 2004US5328	A	20040224	200461	E
EP 1603628	A2	20051214	EP 2004714099	A	20040224	200582	E
			WO 2004US5328	A	20040224		

Priority Applications (no., kind, date): US 2003449263 P 20030224; US 2003449174 P 20030224; US 2004784231 A 20040224

Alerting Abstract US A1
NOVELTY - Protecting a **kidney** (107) in a patient (101) from an insult comprises at least partially **occluding** a **renal** vein, elevating a **renal** vein blood pressure and reducing the **renal** vein blood pressure from the elevated blood pressure.
DESCRIPTION - INDEPENDENT CLAIMS are also included for:
minimizing radiocontrast nephropathy in a patient which comprises at least partially occluding a renal vein and elevating a renal vein blood pressure during a period coinciding with an injection of contrast in the blood, and
a system for treating radiocontrast nephropathy in a patient which comprises a renal catheter (111) comprising a distal tip section having a renal vein occlusion device and a renal vein pressure detector, and a proximal section external of the patient when the distal tip section is positioned in a renal vein, and an actuator for the renal vein occlusion device and connectable to the proximal section of the renal catheter, where the actuator controls the renal vein occlusion device.
USE - Used for protecting a kidney in a patient from an insult, e.g. injections, surgical procedures or hypertension.
ADVANTAGE - The method reduces the exposure of kidneys to insult.
DESCRIPTION OF DRAWINGS - The figure is a schematic diagram of the kidneys and vascular systems in a patient.
101 Patient
107 Kidney
111 Catheter
114 Monitoring console
116 Conduit
Class Codes
International Patent Classification

IPC	Class Level	Scope	Position	Status	Version Date
A61B-005/02; A61F; A61M-027/00			Main		"Version 7"

US Classification, Issued: 600500000
DWPI Class: B05; P31; P34
Manual Codes (CPI/A-N): B11-C04; B14-N10

10/5/2 (Item 2 from file: 350)
Derwent WPIX

0014424697 *Drawing available*
WPI Acc no: 2004-614920/200459
Related WPI Acc No: 2004-634443
XRAM Acc no: C2004-221497
XRPX Acc No: N2004-486237
Protection of kidney in mammalian patient to prevent or treat acute renal failure involves temporarily and reversibly increasing fluid pressure in the renal pelvis or blood pressure in the renal veins
Patent Assignee: PLC SYSTEMS INC (PLCS-N)
Inventor: **GELFAND M; LEVIN H; LEVIN H R**
Patent Family (5 patents, 107 countries)

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20040163655	A1	20040826	US 2003449174	P	20030224	200459	B
			US 2003449263	P	20030224		
			US 2004784807	A	20040224		
WO 2004075948	A2	20040910	WO 2004US5327	A	20040224	200459	E
EP 1599240	A2	20051130	EP 2004714083	A	20040224	200578	E
			WO 2004US5327	A	20040224		

JP 2006518649	W	20060817	WO 2004US5328	A	20040224	200654	E
			JP 2006503812	A	20040224		
JP 2006518758	W	20060817	WO 2004US5327	A	20040224	200654	E
			JP 2006503811	A	20040224		

Priority Applications (no., kind, date): US 2003449263 P 20030224; US 2003449174 P 20030224; US 2004784807 A 20040224

Alerting Abstract US A1

NOVELTY - Protecting the **kidney** in a mammalian patient comprising reducing a **renal** function of at least one **kidney** (107) by artificially increasing pressure in a urinary tract of the **kidney** and reducing the pressure in the urinary tract to increase the **renal** function above the reduced **renal** function. The increase of pressure in the urinary tract is temporary (for at least one hour) and reversible, is new.

DESCRIPTION - The urinary tract pressure is increased by artificially infusing fluid into bladder of the patient prior to administration of a contrast agent or prior to hypotensive surgery.

An INDEPENDENT CLAIM is included for a system for preventing or treating acute **renal** failure (ARF) in mammalian patient.

USE - The method is useful for preventing or treating acute **renal** failure from such causes as radiocontrast nephropathy in a mammalian patient (claimed) and protecting the **kidney** from damage that can cause **renal** ischemia, **renal** medullary hypoxia.

ADVANTAGE - The method allows temporary reduction of the **renal** oxygen demand. The treatment increases **renal** blood flow to increase the ratio of oxygen supply to oxygen demand of the **kidney** by primarily decreasing the demand. The oxygen demand of the **kidney** may be reduced by at least partially, temporarily and reversibly impeding the ability of the **kidney** to filter blood. The **renal** blood flow to glomerular filtration rate (GFR) ratio of **kidneys** is artificially increased for duration of the insult that can last from several hours to weeks. The method prevents impending ARF **renal** failure regardless of etiology; minimizes the damage from existing ARF; reduces hospital days; reduces mortality and morbidity; and reduces cost.

DESCRIPTION OF DRAWINGS - The figure illustrates the treatment of a patient by increasing **renal** vein pressure.

101 Patient
106 **Renal** vein
107 **Kidney**
111 Catheter
112 **Balloon**
114 **Balloon** inflation device.

13/26,TI/1 (Item 1 from file: 350)
Derwent WPIX

0016432527
WPI Acc no: 2007-148725/

Bilateral renal neuromodulation providing method, involves modulating left neural fiber that contributes to renal function of left kidney of patient using applicators positioned within renal vasculature of patient

13/26,TI/2 (Item 2 from file: 350)
Derwent WPIX

0016355506
WPI Acc no: 2007-071676/

Monopolar renal neuromodulation, for treating, e.g. hypertension, comprises electrically coupling ground pad to exterior, and delivering field between electrode and ground pad to modulate function of neural fiber

13/26,TI/3 (Item 3 from file: 350)
Derwent WPIX

0016176221
WPI Acc no: 2006-707861/
Congestive heart failure treating method for patient, involves modulating function of neural fiber that contributes to renal function of patient`s kidney, delivering neuromodulatory agent to fiber, and denervating kidney

13/26,TI/4 (Item 4 from file: 350)
Derwent WPIX

0015656331
WPI Acc no: 2006-220513/
Patient hydration system for preventing radiocontrast nephropathy, has urine collection system, infusion system, and control system having a meter configured to determine urine output and a controller configured to adjust the infusion rate

[File 155] **MEDLINE(R)** 1950-2007/Mar 15
[File 5] **Biosis Previews(R)** 1926-2007/Mar W2
[File 73] **EMBASE** 1974-2007/Mar 20
[File 35] **Dissertation Abs Online** 1861-2007/Feb
[File 65] **Inside Conferences** 1993-2007/Mar 20

Set	Items	Description
S1	2068	S AU=(GELFAND M? OR GELFAND, M?)
S2	1938	S AU=(LEVIN H? OR LEVIN, H?)
S3	15	S S1 AND S2
S4	13	RD (unique items)
S5	**13**	**SORT S4/ALL/PY,A**
S6	40	S RENAL()NEPHROPATHY
S7	1753150	S KIDNEY? ? OR RENAL
S8	471491	S OCCLUD? OR OCCLUS? OR BALLOON? ?
S9	693062	S VEIN? ? OR VENOUS
S10	116600	S INSULT OR NEPHROPATHY
S11	651908	S BLOOD()PRESSURE
S12	1580091	S RADIOCONTRAST OR CONTRAST
S13	3976	S S1:S2 NOT S3
S14	0	S S6 AND S13
S15	258	S S7 AND S13
S16	5	S S10 AND S15
S17	0	S S8 AND S11 AND S15
S18	7	S S12 AND S15
S19	6	S S9 AND S15
S20	17	S S16:S19
S21	11	RD (unique items)
S22	**11**	**SORT S21/ALL/PY,A**
S23	419	S CONTRAST()NEPHROPATHY
S24	0	S S1:S2 AND S23

5/6/3 (Item 3 from file: 5)
16345913 **Biosis No.:** 200100517752
Method and apparatus for treatment of congestive heart failure by improving perfusion of the kidney by infusion of a vasodilator
2001

5/6/4 (Item 4 from file: 5)
17160820 **Biosis No.:** 200300129539
Method and apparatus for treatment of congestive heart failure by improving perfusion of the kidney
2003

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